Case Reports

"Acquired" Left Ventricular-to-Right Atrial Shunt (Gerbode Defect) after Bacterial Endocarditis

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We present, with echocardiographic and intraoperative findings, a rare case of left ventricular-to-right atrial communication (Gerbode defect) after endocarditis associated with Staphylococcus aureus. ***(Tex Heart Inst J 1995;22:100-2)***

Left ventricular-to-right atrial communications are rare types of ventricular septal defect known collectively as the Gerbode defect. These defects are usually congenital, as indicated by Gerbode in his original description of 5 cases in 1958,[1] but very rarely they have been seen after bacterial endocarditis.[2-4] We present the case of a patient with such a defect after an episode of endocarditis associated with *Staphylococcus aureus* infection.

Case Report

In early June of 1993, a 30-year-old patient presented with pyrexia and malaise. Onset had been recent: 2 weeks earlier, in May, he had undergone a complete medical examination, required by his profession, and had been judged normal. At that time, no heart murmur had been heard. In June, a 3/6 holosystolic murmur and a 2/6 diastolic murmur were audible. After admission, he developed complete atrioventricular (AV) block and required pacing for 1 week. Thereafter, 1st degree AV block remained. Blood cultures indicated a *Staphylococcus aureus* infection, and the patient was started on a 6-week regimen of oxacillin administered intravenously. The evident bacterial endocarditis was complicated by transient renal failure requiring hemodialysis. After 3 sessions of dialysis over a 10-day period, kidney function recovered completely.

In November of 1993, the patient was readmitted for further investigation. A transthoracic 2-dimensional echocardiogram (left parasternal view, short axis) showed a defect in the continuity of the root of the aorta, including a membrane in the area of the right sinus of Valsalva, which moved with the cardiac cycle, suggesting a ruptured aneurysm. The transthoracic echocardiogram also demonstrated a bicuspid aortic valve with prolapse and minimal-to-moderate regurgitation, with the regurgitant flow directed towards the interventricular septum. Transthoracic color Doppler echocardiography demonstrated an important left-to-right shunt between the root of the aorta and both the right atrium and the right ventricle. In the apical 4-chamber view, the defect seemed situated at the level of the interventricular septum, just below the aorta, consistent with a subaortic ventricular septal defect. Pulsed Doppler echocardiography showed a left-to-right shunt caused either by ruptured aneurysm of the sinus of Valsalva or by a subaortic ventricular septal defect associated with aortic regurgitation. Catheterization showed a subaortic left-to-right shunt with a Qp/Qs flow ratio of 2.0 and no pulmonary hypertension. The coronary vessels were normal. The aortic valve regurgitation was estimated to be mild to moderate.

In consideration of the significant left-to-right shunt, a decision was made to operate. With the patient anesthetized and intubated, preoperative transesophageal echocardiography was performed on 12 January 1994 with a biplane transducer. In the analysis of the immediate subaortic area (Fig. 1), no improvement

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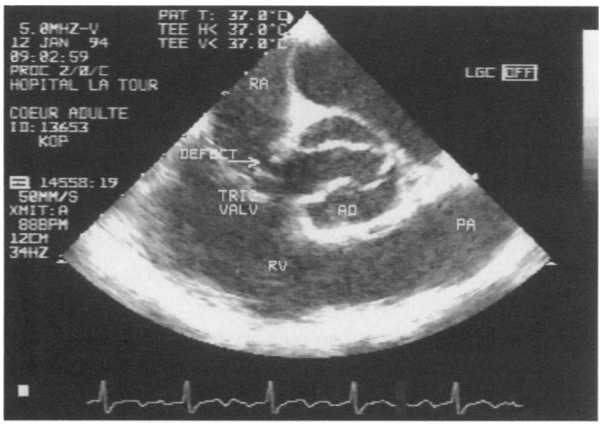

Fig. 1 *Transesophageal echocardiogram showing a subaortic ventricular septal defect with flow both to the right atrium and to the right ventricle.*

AO = aorta; PA = pulmonary artery; RA = right atrium; RV = left atrium

could be seen in comparison to the transthoracic echocardiogram. Doppler showed a systolic and diastolic flow of unknown origin. A more precise diagnosis was rendered difficult by the high subaortic location and by the presence of a prolapsed aortic leaflet moving through the ventricular septal defect with each cycle, which gave the impression of an aneurysmal membrane. Aortic regurgitation with flow directed towards the interventricular septum made interpretation of pulsed and continuous Doppler difficult. Possibly a multiplane transducer would have been useful.

With the patient on cardiopulmonary bypass and with the heart arrested by means of cold crystalline cardioplegia, the right atrium was opened. The fossa ovalis was intact. There was a 12-mm defect just above the septal leaflet of the tricuspid valve, near the septal commissure (Fig. 2). This defect communicated with the left ventricle and with the right ventricle; the septal leaflet of the tricuspid valve was detached from the annulus. The tricuspid valve showed no signs of endocarditis. There were no vegetations on the rims of the defect.

The aorta was opened transversely. There was no defect in the sinus of Valsalva. The aortic valve was bicuspid with no perforations in the leaflets, but with slight thickening and prolapse. The septal defect was just below the aortic annulus.

Repair was performed through the right atriotomy. A small Dacron patch was sutured to the rim of the defect using interrupted Prolene sutures. The tricuspid valve was attached to the Dacron patch (Fig. 3).

After closure of the atriotomy and aortotomy, the patient came off bypass with minimal catecholamine support. He was in complete AV block but had a rapid junctional rhythm. Intraoperative 2-dimensional transesophageal echocardiography showed complete closure of the defect, no tricuspid regurgitation, and unchanged aortic regurgitation.

The patient was extubated the next day. He remained in complete AV block, but had an adequate junctional escape rhythm. His postoperative course was otherwise uneventful. Electrophysiologic testing was delayed for several weeks after discharge, in view of the narrow QRS segment and adequate junctional rate.

Discussion

Septic perforation of the ventricular septum in the course of bacterial endocarditis has been reported rarely in the literature. A Gerbode-type defect (left ventricular-to-right atrial shunt) due to bacterial endocarditis is even more rare; it has been recorded only 3 times to our knowledge.[2-4] Our patient had a thorough physical examination only 2 weeks before endocarditis became symptomatic, and no systolic or diastolic murmur was noted. It is exceedingly un-

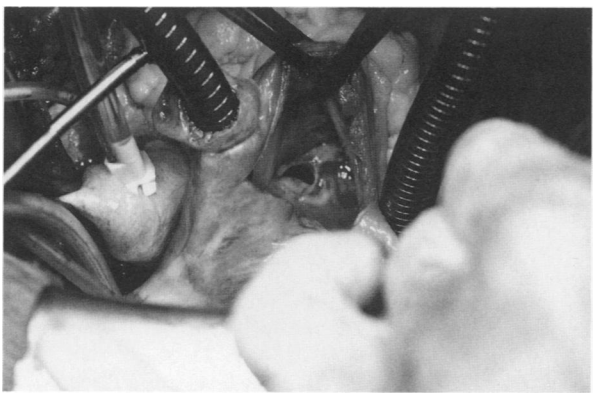

Fig. 2 *Right atrial view of the communication between the left ventricle and the right atrium. The communication is below the septal leaflet of the tricuspid valve.*

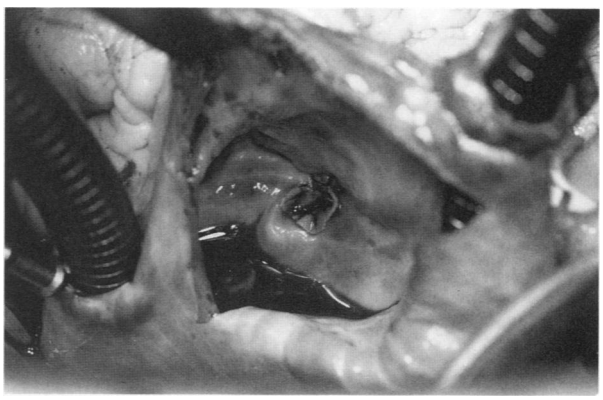

Fig. 3 *Right atrial view of the defect after closure with a small Dacron patch.*

likely that the murmur was present and went unnoticed. The probability of a bacterial origin of the defect is high in this case.

Riemenschneider and Moss[5] have described 2 types of left ventricular-to-right atrial communication: supravalvular and infravalvular, depending on whether the defect in the membranous septum is above or below the tricuspid valve. Our patient's defect was of the supravalvular type.

Echocardiographic interpretation of this patient's condition was difficult because of the associated aortic valve prolapse; even intraoperative 2-dimensional transesophageal echocardiography with pulsed Doppler could not render the diagnosis certain. Preoperative diagnoses of left ventricular-to-right atrial communication in adults have been made, but have been infrequent.[6]

Apart from the diagnostic problem that this case presented, it raises the interesting and unanswered question of the mechanism of septal defect creation in bacterial endocarditis. Lesions of endocarditis are most often seen on valve leaflets or on valve annuli and most often on malformed (i.e., bicuspid) or previously diseased (i.e., after rheumatic disease) valves. Although ventricular septal defects have been described after bacterial endocarditis, the mechanism of their creation has not been elucidated in the 3 published cases of "acquired" Gerbode syndrome, in the publication of a recent clinicopathologic conference,[7] or in any other source that has come to our attention.

Therefore, we can only hypothesize about the mechanism of the septal perforation in our patient. It is possible that his perimembranous ventricular septal defect was congenital in origin and had closed years earlier by any of several mechanisms suggested by Anderson and colleagues.[8] The mechanism most often implicated is redundant tricuspid tissue that closes the defect when a septal aneurysm forms. Residual septal aneurysms could then perforate secondarily as a result of bacterial invasion, giving rise to the condition described. Although bacterial invasion of an intact septum is possible, it is highly unlikely; invasion of any congenital anomaly of the septum is more plausible. In our patient, transient complete AV block during the episode of endocarditis indicates infection in the area of the bundle of His and dismisses, in our minds, the possibility of a patent ventricular septal defect overlooked prior to the episode of endocarditis.

Finally, it is interesting to consider whether minor abnormalities of the ventricular septum, if detected, would entail lifelong antibiotic prophylaxis for endocarditis and whether such prophylaxis would have prevented the lesion described in this report.

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